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<u>L3</u>	L2 and (pulmonary adj1 infection)	29	<u>L3</u>
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L3: Entry 27 of 29

File: USPT

Jun 17, 1997

DOCUMENT-IDENTIFIER: US 5639661 A

TITLE: Genes and proteins for treating cystic fibrosis

Brief Summary Text (7):

Sequence analysis of the CFTR gene of CF chromosomes has revealed a variety of disease causing mutations (Cutting, G. R. et al. (1990) Nature 346:366-369; Dean, M. et al. (1990) Cell 61:863:870; and Kerem, B-S. et al. (1989) Science 245:1073-1080; Kerem, B-S et al. (1990) Proc. Natl. Acad. Sci. USA 87:8447-8451). Population studies have indicated that the most common CF mutation, a deletion of the 3 nucleotides that encode phenylalanine at position 508 of the CFTR amino acid sequence (.DELTA.F508), is associated with approximately 70% of the cases of cystic fibrosis. This mutation results in the failure of an epithelial cell chloride channel to respond to cAMP (Frizzell R. A. et al. (1986) Science 233:558-560; Welsh, M. J. (1986) Science 232:1648-1650.; Li, M. et al. (1988) Nature 331:358-360; Quinton, P. M. (1989) Clin. Chem. 35:726-730). In airway cells, this leads to an imbalance in ion and fluid transport. It is widely believed that this causes abnormal mucus secretion, and ultimately results in pulmonary infection and epithelial cell damage.

Brief Summary Text (13):

A second approach to curing cystic fibrosis, "protein replacement" seeks to deliver functional, recombinant CFTR to CF mutant cells to directly augment the missing CFTR activity. The concept of protein replacement therapy for CF is simple: a preparation of highly purified recombinant CFTR formulated in some fusogenic liposome or reassembled virus carrier delivered to the airways by instillation or aerosol. However, attempts at formulating a CF protein replacement therapeutic have met with difficulties. For example, CFTR is not a soluble protein of the type that has been used for previous protein replacement therapies or for other therapeutic uses. There may be a limit to the amount of a membrane protein with biochemical activity that can be expressed in a recombinant cell. There are reports in the literature of 10.sup.5 -10.sup.6 molecules/cell representing the upper limit (H-Y Wang et. al J. Biol. Chem 264:14424 (1989)), compared to 2000 molecules/second/cell being reported for secreted proteins such as EPO, insulin, growth hormone, and tPA.

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L3: Entry 24 of 29

File: USPT

May 26, 1998

DOCUMENT-IDENTIFIER: US 5756353 A

**\*\* See image for Certificate of Correction \*\***

TITLE: Expression of cloned genes in the lung by aerosol-and liposome-based delivery

Detailed Description Text (28):

Similarly, genes coding for peptides known to display antiviral and/or antibacterial activity, or stimulate the host's immune system, can also be administered to the lung in order to treat pulmonary infections. Thus, the genes encoding many of the various cytokines (or functional fragments thereof), such as the interleukins, interferons, and colony stimulating factors, will find use with the instant invention. The gene sequences for a number of these substances are known. It has been found that recombinant interferon- $\gamma$ , when administered by aerosol, retains its activity and displays little or no toxicity in mammals. Debs, et al., J. Immunol. (1988) 140:3482-3488. Furthermore, aerosolized interferon- $\gamma$  produces significant anti-pneumocystis carinii pneumonial (PCP) activity in immunodeficient mice with PCP. Beck et al., Infect. and Immun. (1991)

Detailed Description Text (67):

To treat pulmonary infections such as bronchitis and pneumonia, it will usually be necessary to administer at least one dose per day over a period of about 4 to about 21 consecutive days or longer. The treatment is usually carried out on consecutive days because new areas of the lungs open up to penetration and deposition of the nucleic acid with increasing resolution of the infection. The success of the treatment can be monitored and the administration regimen altered by assessing conventional clinical criteria; e.g., clearing of radiographic infiltrate, improved arterial PO<sub>2</sub> (e.g., >70 mmHg), reduction in dyspnea, respiratory rate and/or fever.

Detailed Description Text (68):

For the treatment of genetic disorders, such as cystic fibrosis, the liposome-nucleic acid complex will be administered at regular intervals, from once a week to once every one to several months, in order to replace the normal CFTR protein in critical host airway cells, since these cells continue to turn over. It may also be possible to stably transfect the CFTR gene into appropriate lung stem cells, which would then provide a continuous source of normal airway cells without requiring lifelong treatment.

Other Reference Publication (5):

Alton, E., et al. (1993) "Non-invasive liposome-mediated gene delivery can correct the ion transport defect in cystic fibrosis mutant mice", Nature Genetics, 5:135-142.

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File: USPT

Sep 21, 1999

DOCUMENT-IDENTIFIER: US 5955442 A

TITLE: Methods for treating respiratory disease

Brief Summary Text (5):

Treatment of pulmonary diseases generally requires antibiotic therapy which is frequently ineffective. Recently, however, cystic fibrosis has been treated using DNase. The rationale for such therapy is that degrading DNA in sputum reduces the viscosity of the sputum and results in an increased ability of the patient to evacuate sputum from the lungs and nasal passages. However, no known report advocates using DNA itself as a treatment for any pulmonary infection.

Other Reference Publication (8):

Alton, E.W.F.W. et al., "Noninvasive liposome-mediated gene delivery can correct the ion transport defect in cystic fibrosis mutant mice," Chemical Abstracts, 119 (21):62 (Nov. 22, 1993) (Abstract 217089w).

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File: USPT

Jun 12, 2001

DOCUMENT-IDENTIFIER: US 6245735 B1

TITLE: Methods and products for treating pseudomonas infection

Brief Summary Text (19):

The foregoing covalent conjugates are useful in delivering bioactive agents to cells and/or tissues expressing a CFTR. Thus, methods are provided for delivering a bioactive agent to a tissue expressing a cystic fibrosis transmembrane conductance regulator to treat a condition susceptible to treatment by the bioactive agent. A bioactive agent coupled to a polysaccharide is administered to a subject in need of such treatment, in an amount effective for treating the condition. The polysaccharide is as described above. The bioactive agent can be noncovalently or covalently linked to the polysaccharide, or the bioactive agent can be contained in a liposome comprising a lipid biocompatible with a human subject, wherein the polysaccharide is covalently coupled to the lipid.

Detailed Description Text (60):

16. Tang H, Kays M, Prince A. Role of *P. aeruginosa* pili in acute pulmonary infection. Infect Immun. 63:1278-1285; 1995.

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File: USPT

Jun 4, 2002

DOCUMENT-IDENTIFIER: US 6399585 B1

TITLE: In Utero treatment of CFTR-related deficiencies

Brief Summary Text (23):

(Stern 1997) has roughly correlated the amount of functional CFTR produced and the phenotype. Pancreatic exocrine deficiency is at one extreme, with less than 1% of normal CFTR function, followed by progressive pulmonary infection (<4.5%); demonstrable sweat abnormality (<5%); congenital absence of vas deferens; no known abnormality (>10%).

Brief Summary Text (80):

Twenty clinical trials of gene therapy for cystic fibrosis have been initiated using viral and non-viral vectors for gene transfer (Marcel and Grausz 1997). Vectors that have been studied in attempts to develop gene therapy for CF include adenoviruses, adeno-associated viruses (AAV), and liposomes (MacVinish, Goddard et al 1997).

Brief Summary Text (86):

DOTAP cationic liposome mediated CFTR gene transfer to the nasal epithelium of patients with cystic fibrosis has been shown to be safe and efficacious (Porteous, Dorin et al. 1997).

Brief Summary Text (116):

One mode of the present invention comprises in utero treatment of an animal to temporarily mitigate the condition of CFTR deficiency by transgenic therapy, with the surprising result that the animal subsequently is relieved of many or all of the symptoms of cystic fibrosis. The treatment may comprise transfer of the CFTR gene by viral vector, liposome or other medium. Many examples of methods of genetic transfer are mentioned herein, and any of these as well as other methods of gene transfer not specifically mentioned, can be used to implement in utero gene therapy with long-lasting improvement on the symptoms of cystic fibrosis.

Detailed Description Text (6):

Multiple organ systems are affected in CF disease. However, the most lethal pathology is the mucous plugging, chronic inflammation, and bronchiectasis that result in respiratory failure and cor pulmonale (12). Although thickened mucous secretions can be explained by defective secretion of lung liquid and hyperabsorption of sodium by the respiratory epithelium, many of the symptoms of the disease cannot be explained by CFTR's chloride channel function alone. The association of CF with chronic pulmonary infections due to *Pseudomonas aeruginosa* as well as other organisms remains particularly puzzling (13,14,15,16).

Detailed Description Text (388):

141 Porteous, D. J., J. R. Dorin, et al. (1997). "Evidence for safety and efficacy of DOTAP cationic liposome mediated CFTR gene transfer to the nasal epithelium of patients with cystic fibrosis." Gene Ther 4(3): 210-8.

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